NOTES
BRAIN ISCHEMIA

GENERALLY, WHAT IS IT?

PATHOLOGY & CAUSES

- Impaired brain function due to lack of blood

TYPES

Focal ischemia

- Occlusion of blood vessel → ↓ perfusion → affected regions damaged
- ↓ oxygen → ischemic stroke
- Blood vessel rupture → hemorrhagic stroke; bleeding inside parenchyma/between brain membranes

Global ischemia

- Cardiac arrest → whole brain hypoperfusion → brain damage

CAUSES

- Atherosclerotic plaque/thrombosis/emboli
- Hypertension
- Blood vessel malformation
- Cardiac arrest, tachycardia, congenital heart problems
- Tumors

SIGNS & SYMPTOMS

- Altered consciousness; weakness; problems with vision, hearing, swallowing; dizziness, vertigo
- See mnemonic for common symptoms

DIAGNOSIS

DIAGNOSTIC IMAGING

CT scan

- Visualize trauma, bleeding, skull fracture

MRI

- Visualize hypointense, hyperintense blood clot

MR/CT angiography

- Visualize occlusions, aneurysms

TREATMENT

MEDICATIONS

- Antiplatelet medications (e.g. aspirin/clopidogrel)
- IV tissue plasminogen activator (tPA)
- Mannitol, other osmotic diuretics
  - ↑ intracranial pressure treatment

SURGERY

- Evacuation of blood clot
- ↑ intracranial pressure treatment

OTHER INTERVENTIONS

- Manage conditions that worsen prognosis (e.g. hyperglycemia, fever)

MNEMONIC: FAST

Common stroke symptoms

Facial drooping

Arm weakness

Speech difficulties

Time: reminder to call emergency services

EPIDURAL HEMATOMA

osms.it/epidural_hematoma

PATHOLOGY & CAUSES

- Nervous tissue compression due to accumulation of blood in epidural space
- Head trauma → skull fracture → damage of blood vessels through dura mater → extradural blood accumulation → rapid, limited expansion of hematoma due to tight dura adherence at cranial sutures → brain tissue compression → neurological decline

TYPES

Intracranial

- Frontal injuries
  - Anterior ethmoidal artery
- Temporoparietal (most common)
  - Middle meningeal artery
- Occipital
  - Transverse, sigmoid sinus
- Vertex
  - Superior sagittal sinus

Spinal

- Venous plexus of lumbar, thoracic regions

CAUSES

- Neurosurgical procedures complication
- Trauma

Intracranial epidural hematoma

- Head trauma → pterion skull fracture (most common)
- Blood vessel malformations

Spinal epidural hematoma

- Trauma (e.g. lumbar puncture/epidural anesthesia)

RISK FACTORS

- More common in individuals who are biologically male, between 2–60 years
- Pregnancy
  - Spontaneous spinal hematoma (very rare)
- Systemic lupus erythematosus
  - Vasculitis, associated with immune system reaction
- Coagulopathies, bleeding diathesis, sickle cell anemia

COMPLICATIONS

- ↑ intracranial pressure
- Supratentorial herniation → compression of arteries → ischemic stroke
- Infratentorial herniation → brainstem compression → heart, respiratory arrest
- Paralysis/sensory loss
- Seizures

SIGNS & SYMPTOMS

- Initial loss of consciousness, lucid state if blood slowly accumulating; delayed neurological deterioration consequence of enlarging hematoma compression
- Intracranial epidural hematoma
  - Broken skull with hematoma
  - Otorrhea/rhinorrhea
  - Altered consciousness
- ↑ intracranial pressure
  - Headache
  - Nausea with vomiting
  - Cushing reflex (↑ blood pressure, ↓ heart rate, irregular breathing)
  - Focal signs (weakness of extremities on opposite side; dilated pupil on injured side due to compression of CN III)
- Spinal epidural hematoma
  - Radicular back pain (resembles pain from herniated discus)
  - Sensory defects
  - Urinary, fecal incontinence

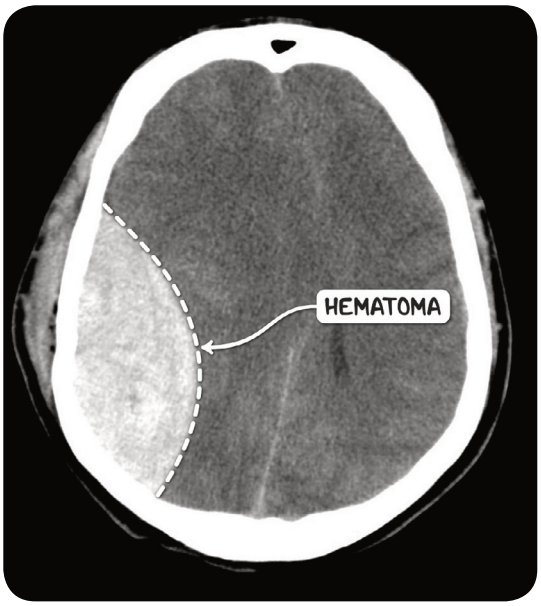

Figure 64.1 A CT scan of the head in the axial plane demonstrating a large epidural hematoma with a classical biconvex shape.

DIAGNOSIS

DIAGNOSTIC IMAGING

X-ray

- Skull fracture

CT scan

- *Hematoma:* typically presents as a biconvex, relatively heterogeneous, high density mass in the space between skull, brain; does not cross sutures
- *Swirl sign:* bleeding into blood clot, diverse hypoattenuated foci
- Assess hematoma volume
- Skull fracture

MRI

- *T2-WI:* acutely
  - Hypointense blood clot due to deoxyhemoglobin
- *T1, T2-WI:* in following weeks
  - Deoxy → methemoglobin; hyperintense blood clot
- *T1-WI:* months later
  - Methemoglobin → hemosiderin; hypointense mass

TREATMENT

MEDICATIONS

- Mannitol, other osmotic diuretics
  - ↑ urine excretion, ↓ intracranial pressure
- Anticoagulation reversal
  - Individuals undergoing surgery, on anticoagulation therapy

SURGERY

- Craniotomy
  - Evacuation of blood mass
- Embolization/ligation of damaged blood vessel
- Trephination (burr-hole)
  - In acute EDH, if neurosurgical procedure delayed
- Laminectomy
  - ↓ blood in spinal epidural hematoma

OTHER INTERVENTIONS

- Observation, nonoperative management
  - Awake, conscious individuals
  - If hematoma volume < $30cm^3$, thickness < 15mm, midline shift < 5mm

INTRACEREBRAL HEMORRHAGE

osms.it/intracerebral-hemorrhage

PATHOLOGY & CAUSES

- Condition characterized by blood vessels rupture → intraparenchymal blood accumulation
- Blood vessel trauma, rupture → creates pool of blood → tissue, surrounding blood vessel compression → hypoxia in downstream tissue → damage due to compression, oxygen lack

CAUSES

Hypertension

- Most common
- Leads to
  - Atherosclerosis in large arteries
  - Hyaline arteriolosclerosis → focal arterioles necrosis → small wall ruptures → subclinical microbleeds
  - Charcot–Bouchard aneurysms (microaneurysms)
- Basal ganglia; thalamus; midbrain; pons; cerebellum primarily affected

Vascular abnormalities

- Cerebral amyloid angiopathy
  - Deposition of amyloid in blood vessel walls → vessels more prone to rupture
  - *Lobar localization:* parietal, occipital lobes
  - *Blood vessels:* leptomeningeal, cerebral cortical arterioles
- Arteriovenous malformations
  - Usually affect children
- Aneurysm, vasculitis, vascular tumours (e.g. hemangioma)

Other causes

- Secondary to ischemic stroke
  - Blood flow blockage → reperfusion → ↑ chance of blood vessel rupture → bleeding into dead tissue (hemorrhagic conversion)
- Posttraumatic
- Coagulopathies
- Sickle cell disease

RISK FACTORS

- Individuals who are biologically male of Asian descent
- Black individuals who are biologically male of African descent
- Heavy alcohol use; amphetamines, cocaine abuse, antithrombotic medications; ↓ LDL, cholesterol, triglycerides; previous cerebrovascular insult

COMPLICATIONS

- Hemorrhage enlargement
  - In hemorrhage border
  - Poor prognosis
- Intraventricular, subarachnoid expansion
- Hydrocephalus

SIGNS & SYMPTOMS

- Begin slowly, worsen gradually
- Enlargement of hematoma within few hours, ↑ intracranial pressure
  - Altered consciousness, headache, nausea, vomiting, unequal pupil size
- Fever

Area of brain affected

- *Anterior/middle cerebral artery:* numbness, sudden muscle weakness
- *Posterior cerebral artery:* impaired vision
- *Broca's area:* slurred speech
- *Wernicke's area:* difficulty understanding speech

Focal neurological signs

- *Basal ganglia manifestation:* loss of contralateral sensory, motor functions; homonymous hemianopsia
- *Thalamus:* contralateral loss of

sensory, motor functions; homonymous hemianopsia; aphasia if dominant/neglect if nondominant; narrowed pupils unreactive to light
- *Lobar manifestation:* homonymous hemianopsia; if frontal region, contralateral leg plegia/paresis; seizures
- *Pons:* coma within few minutes of hemorrhage; quadriplegia, miosis/deafness; speaking difficulties when awake
- *Cerebellum:* ataxia; same side face weakness; loss of face, body sensory function; occipital headache, neck stiffness

DIAGNOSIS

DIAGNOSTIC IMAGING

CT scan
- Hyperdense blood mass acutely; isodense, ring enhancement appearance in subsequent weeks; hypodense chronically
- Trauma
  - Multifocal bleedings
- Coagulum retracts, edema develops
  - Confused with hemorrhagic infarction

CT angiography
- *Spot sign:* unifocal/multifocal enhancement of contrast; ↑ risk of hematoma expansion

MRI (T2-WI)
- Hyperacute (first 24 hours)
  - Hyperintense center of mass
  - Hypointense periphery, border
- Subacute
  - *Hypointense in > three days:* intracellular methemoglobin
  - *Hyperintense in > seven days:* lysis of red blood cells; extracellular methemoglobin
  - *Chronic:* hypointense; after two weeks

MR angiography
- Vasculitis, arteriovenous malformations, other blood vessel pathology

LAB RESULTS
- Prothrombin time (PT), activated partial thromboplastin time (aPTT), platelet count
  - If cause for bleeding diathesis unclear

TREATMENT

MEDICATIONS
- Vitamin K, unactivated prothrombin
  - With anticoagulant usage
- Protamine sulfate
  - For heparin users
- Antipyretics
  - Fever reduction
- Osmotic diuretics (e.g. mannitol)
  - Regulation of ↑ ICP
- Saline
  - Fluid replacement
- Nicardipine/nitroprusside/enalapril/nitroglycerin
  - Hypertension
- Phenytoin/levetiracetam
  - Seizures

SURGERY
- Ventriculostomy
  - Regulation of ↑ intracranial pressure
- If hemorrhage > 3cm/1.2in/lobar of young persons/brainstem compression
  - Craniotomy with clot removal
  - Stereotactic aspiration
  - Endoscopic evacuation

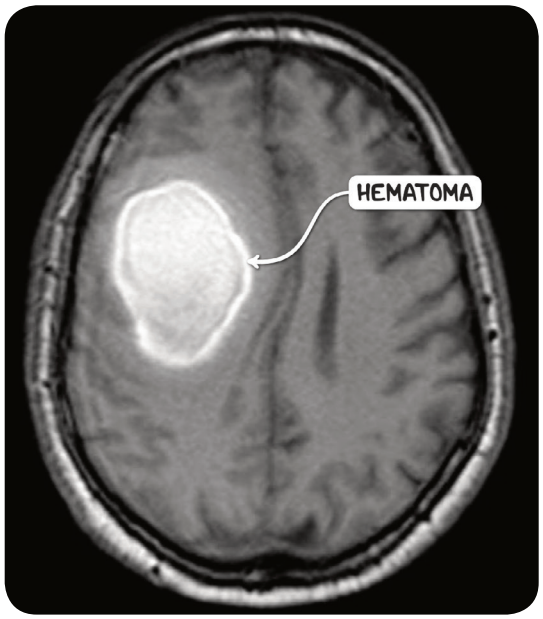

Figure 64.2 A CT scan of the head in the axial plane demonstrating a right-sided, periventricular, intracerebral hemorrhage.

ISCHEMIC STROKE

osms.it/ischemic-stroke

PATHOLOGY & CAUSES

- Decreased blood supply in specific brain area due to blood vessel obstruction → hypoperfusion, tissue hypoxia, infarction
- ↓ blood flow → lack of oxygen, glucose in brain → ↓ adenosine triphosphate (ATP) production, electrochemical gradient → cell death

Two mechanisms of cell death

- *Sodium buildup:* water follows sodium → cell swelling, death
- *Calcium buildup:* creates oxygen radicals → damages mitochondrial, lysosomal lipid membrane → seeping of degradative enzymes, apoptosis-inducing factors → cell death

Two zones

- Ischemic core
  - Brain tissue dies from ischemia within few minutes of stroke
  - Blood flow < 10ml/100g tissue/minute
- Ischemic penumbra
  - Periphery of affected region preserved due to collateral circulation; chance of survival if blood restored quickly
  - Blood flow < 25ml/100g tissue/minute
  - Infarction zone spreads if blood supply not restored quickly

TYPES

Five subtypes (TOAST classification)

- Large artery atherosclerosis
- Small artery strokes
- Cardioembolic infarction
  - Formation of emboli in heart → lodging in brain arteries
- Other determined pathology
- Undetermined pathology

CAUSES

Thrombosis

- May lead to obstruction inside blood vessel
- Narrowing of blood vessel due to atherosclerotic plaque → gradual ↓ blood flow
- Damage to atherosclerotic fibrous cap → platelet, clotting cascade activation → thrombus formation with sudden stop of blood flow

Embolism

- Four classes based on emboli origin
  - *Cardiac emboli:* atrial fibrillation, rheumatic valve disease, infective endocarditis, dilated cardiomyopathies, left atrial myxoma
  - *Possible cardiac/aortic emboli:* calcification of mitral valve annulus, patent foramen ovale, atheroma in ascending/arch of aorta, atrial septal aneurysm
  - *Arterial emboli:* detachment of blood clot (e.g. atherosclerotic plaque in bigger upstream artery) → emboli travels through blood → lodges in smaller downstream artery
  - *Cryptogenic:* unknown origin of emboli

Lacunar infarct

- Affects small blood vessels of distal vertebral, basilar artery, middle cerebral artery, circle of Willis
  - *Lipohyalinosis:* buildup of hyaline in arterioles wall → hypertrophy of tunica media → progressive narrowing of arterioles until blood flow stops
  - *Microatheromatoma:* narrowing of blood vessel due to debris accumulation within wall

Hypoperfusion

- Lack of blood reaching brain due heart failure, ↓ cardiac output
- Most vulnerable
  - Spaces between supply of two arteries (watershed regions)

Inflammation of blood vessel wall

- E.g. Takayasu/giant cell arteritis

Moyamoya disease

- Progressive stenosis of cerebral arteries → ischemia

Dissection of artery wall

RISK FACTORS

- Age (esp. > 55)
- More common in individuals who are biologically male
- More common in black individuals of African descent
- Migraine headaches with aura
- Genetics; specific gene loci associated with stroke subtypes
  - *ABO loci:* all subtypes
  - *HDAC9:* large vessel stroke
  - *PITX2, ZFHX3:* cardioembolic stroke
- Hematologic disorders
  - Multiple myeloma, sickle cell disease, polycythemia vera; esp. in younger individuals
- Hypertension, diabetes mellitus, heart diseases, dyslipidemia, hyperhomocysteinemia, smoking, physical inactivity, cocaine abuse

COMPLICATIONS

- Blood reaches infarcted regions through collateral blood vessel/dissolution of occlusive embolus/thrombus; first week

Hemorrhagic transformation

- Ischemia → impaired cellular, metabolic functions of affected region; ↑ permeability of damaged blood vessels → resolved cause of ischemia → restored blood flow → blood extravasation
- Gray matter more commonly affected; large number of collateral vessels worsen reperfusion injury
- Massive cerebral infarction; hyperglycemia; ↓ cholesterol, LDL, IV recombinant tissue plasminogen activator (rtPA): higher risk of hemorrhagic transformation

Cerebral edema

- ↑ intracranial pressure with possible herniation
  - *Cytotoxic:* defective ATP pump, swelling of brain cellular elements due to water

accumulation
    - *Vasogenic:* ↑ permeability of blood-brain barrier → ↑ extracellular fluid volume due to ↑ passing of proteins, other macromolecules

Liquefactive necrosis
- *First 48 hours:* edema, paleness of affected region
- *2–10 days:* affected area gelatinous; noticeable border between healthy, damaged tissue
- *3–21 days:* liquefaction of tissue; fluid-filled cavity

Seizures
- Brain injury → ↑ irritability of nervous tissue with neuronal discharges

Deep vein thrombosis
- Esp. immobilized individuals

Pneumonia
- Swallowing mechanism impairment → aspiration pneumonia
- Intubation/ventilatory support → ↑ risk for pneumonia

Dysphagia
- Due to damage of cortex, subcortical structures responsible for swallowing

Dementia
- Altered memory, cognition, behavior due to brain damage

SIGNS & SYMPTOMS

Thrombosis
- Neurological defects

Embolism
- Sudden start of symptoms; maximum defects

Lacunar stroke
- Contralateral, mostly motor/sensory defects; four syndromes
    - *Pure motor stroke:* internal capsule lesion
    - *Pure sensory stroke:* thalamus lesion
    - *Ataxic hemiparesis*
    - *Dysarthria:* speech, swallowing difficulties; facial weakness; hand weakness, clumsiness (clumsy hand syndrome)

Anterior cerebral artery
- Contralateral hemiparesis (esp. leg, face), sensory deficit; inability to understand, produce speech (left hemisphere); impaired judgment; incontinency

Middle cerebral artery
- Contralateral paresis (esp. face, arm), sensory deficit; inability to understand, produce speech (left hemisphere); hemispatial neglect (right hemisphere); homonymous hemianopsia; deviation of eye to damaged side

Posterior cerebellar artery
- Homonymous hemianopsia
- Cortical blindness (bilateral lesion)
- Midbrain
    - Oculomotor, trochlear palsy → dilated pupil
- Thalamus
    - Sensory loss, impaired memory, altered consciousness
- Posterior cerebellar artery syndrome (PICA)
    - AKA “Wallenberg” syndrome
    - Dizziness, nystagmus; speech, swallowing difficulties
    - *Ipsilateral:* facial sensory loss, Horner’s sign, ataxia
    - *Contralateral:* loss of pain, temperature sensation in limbs

Basilar, vertebral arteries
- Dizziness; gait, vision disorders; dysarthria, dysphagia
- Locked-in syndrome
    - Thrombosis/embolism of basilar artery
    - Plegia of head, body muscles, except eye; only blinking, vertical eye movement possible

DIAGNOSIS

DIAGNOSTIC IMAGING
- If applicable

Noncontrast CT scan
- First few hours

- Affected tissue appears normal
- Later
  - Loss of differentiation between white, grey matter
  - Hypodense parenchyma with sulcal effacement
  - Loss of insular ribbon sign

CT perfusion

- Detection of core, ischemic penumbra

CT angiography

- Find thrombus, embolus in blood vessel/intra-arterial thrombolysis

MRI

- T1, T2 weighted imaging (see table)
- Diffusion-weighted imaging
  - Shows ischemic stroke early; differentiation from acute, chronic
- Fluid-attenuated inversion recovery (FLAIR) sequence
  - Hyperintense signal within 12 hours

Transcranial Doppler ultrasound

- Visualization of occlusion in middle cerebral artery/intracranial carotid/vertebrobasilar artery

Conventional angiography

- Visualize occlusion; for confirmation of CTA, MRA findings

LAB RESULTS

- Blood tests
  - ↑ cardiac markers in heart disease
  - ↑ erythrocytes in polycythemia vera
  - Toxicology screening (individual suspected of sympathomimetics abuse)
  - ↑ blood glucose level

OTHER DIAGNOSTICS

- Symptoms, neurological changes scoring
- Based on National Institute of Health stroke scale (NIHSS)

ECG

- Detection of myocardial ischemia/atrial fibrillation

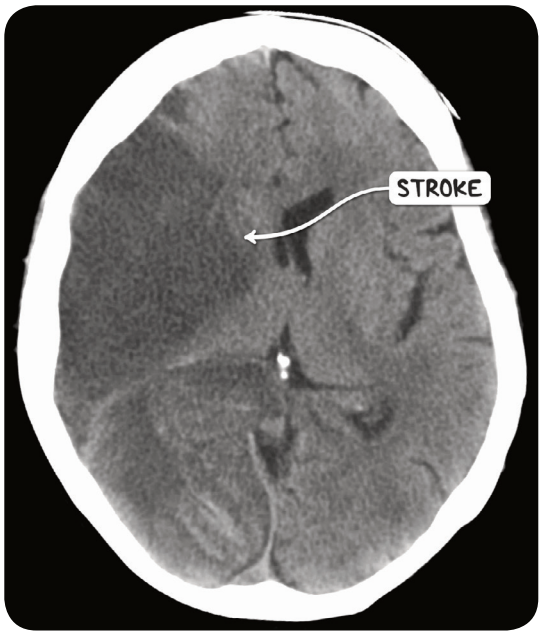

Figure 64.3 A CT scan of the head in the axial plane demonstrating a large ischemic stroke in territory of the middle cerebral artery. The scan was performed three days after the onset of symptoms.

TREATMENT

MEDICATIONS

- Establishment of blood flow in ischemic penumbra
  - *Thrombolytic enzymes:* rtPA; alteplase given within 4.5 hours; after hemorrhagic stroke ruled out
  - *Antiplatelet therapy:* aspirin (325mg orally within 48 hours); other drugs (e.g. clopidogrel/aggrenox)
- Hypertension treatment
  - *IV labetalol/nicardipine:* only if systolic pressure > 220, diastolic > 120 mmHg; except in individuals with vital indications for lowering blood pressure (acute myocardial infarction, kidney failure, dissection of aorta)
- Cerebral edema management
  - *Antipyretic:* if temperature ≥ 40°C/100.4°F
  - *IV insulin:* hyperglycemia; keep glucose between 140–180 mg/dl (7.8–10 mmol/L)

- Prevention
  - *Anticoagulant medications:* emboli prevention (e.g. warfarin, aspirin)

SURGERY

- Establishment of blood flow in ischemic penumbra
  - Mechanical embolus removal in cerebral ischemia (MERCI) retriever
  - Penumbra system (aspiration, extraction)
  - Solitaire revascularization device, Trevo (stent-retriever systems)
- Cerebral edema management
  - Craniectomy

PSYCHOTHERAPY

- If applicable
- Type of psychotherapy (e.g. group therapy, exposure therapy) with goal of psychotherapy

OTHER INTERVENTIONS

- Cerebral edema management
  - *Hyperbaric oxygen therapy:* ↑ pure oxygen supply in damaged regions
  - *Fluid management:* isotonic saline without dextrose
- Protection of airwaves, prevention of aspiration
  - Head elevation by 30%, nothing by mouth/*nil per os* (NPO) status
- Prevention
  - *Control risk factors (for atherosclerosis):* e.g. smoking, hypertension, diabetes, aspirin use; carotid endarterectomy
  - *Lifestyle alteration:* exercising, appropriate diet

MRI FOR ISCHEMIC STROKE

	T1 WEIGHTED IMAGING (T1-WI)	T2 WEIGHTED IMAGING (T2-WI)	NOTES
HYPERACUTE	Hypotense signal (16-24 hours)	Absent flow void signal (2 hours); Hyperintense signal from affected region (8 hours)	
ACUTE (1-7 DAYS)	Hypointense	Hyperintense	T1, T2-WI masked by reperfusion in 24-48 hours → fogging phenomenon
SUBACUTE (7-21 DAYS)	Hypointense	Hyperintense	
CHRONC (> 21 DAYS)	Hypointense	Hyperintense	

SACCULAR ANEURYSM

osms.it/saccular-aneurysm

PATHOLOGY & CAUSES

- Asymmetrical ballooning of blood vessel wall
- Bifurcation of arteries common place esp. on circle of Willis due to weakness of wall, turbulent blood flow
  - Anterior communicating (most common); posterior communicating; middle cerebral; internal carotid; basilar artery tip

TYPES

Type A

- Blood vessel wall with endothelium, linear smooth muscle

Type B

- Disorganization of smooth muscle, thickening of wall

Type C

- Hypocellular wall with thickening of intima/luminal thrombosis

Type D

- Hypocellular wall coated with thin thrombosis layer

CAUSES

- Inborn defect of arteries, lack of external lamina, tunica media → hemodynamic stress over years → gradual ballooning of blood vessel wall, thickening of intima, adventitia

RISK FACTORS

- More common in individuals who are biologically female, > 50 years (due to estrogen deficiency)
- Diseases associated with aneurysm
  - Ehler–Danlos syndrome, pseudoxanthoma elasticum, lupus, autosomal dominant polycystic kidney disease (ADPKD), bacterial endocarditis, fibromuscular dysplasia
- Familial predisposition; smoking; alcohol, cocaine use; hypertension; trauma

COMPLICATIONS

- Warning leaks
  - May precede aneurysm rupture; strong headaches, photophobia, nausea/vomiting
- Rupture → subarachnoid hemorrhage
  - Apex of aneurysm/atheromatous plaque edge
  - ↑ risk in smokers, individuals with migraines, elderly, affection of posterior circulation, larger size
- Ischemia
  - Thrombus forms within aneurysm → detachment of small particles (emboli) → emboli lodges → ischemia of downstream tissue
- Multiple aneurysms

SIGNS & SYMPTOMS

- May be asymptomatic if small
- Mass effect symptoms due to size
  - *Anterior communicating artery:* both leg weakness with positive Babinski sign
  - *Posterior communicating, internal carotid artery:* headaches with palsy of oculomotor nerve
  - *Left middle cerebral artery:* inability to understand, produce speech
  - *Right middle cerebral artery:* contralateral field vision loss

DIAGNOSIS

DIAGNOSTIC IMAGING

CT/MR angiography

- Detect aneurysms > 2mm

TREATMENT

SURGERY

- Endovascular management
  - Aneurysmal coiling with thrombosis → endothelialization across aneurysm neck → prevents rebleeding, regrowth
  - In development: stent-assisted, balloon-assisted coiling; disruptors, flow diverters
- Surgical clipping

OTHER INTERVENTIONS

- Regular monitoring with CTA/MRA
- Avoid smoking, alcohol, drugs, excessive strain

SUBARACHNOID HEMORRHAGE (SAH)

osms.it/subarachnoid-hemorrhage

PATHOLOGY & CAUSES

- Bleeding into space between pia mater, arachnoid membrane
- Injury/spontaneous event → rupture of blood vessel in subarachnoid space → release of blood into cerebrospinal fluid (CSF) → rapid ↑ intracranial pressure

CAUSES

- *Traumatic:* head injury (e.g. bridging vein tear)
- *Spontaneous:* arterial origin (more common)
  - Rupture of saccular "berry" aneurysms (e.g. anterior half of circle of Willis)
  - Arteriovenous blood vessel malformations

RISK FACTORS

- More common in individuals who are biologically female, elderly
- Smoking; hypertension; alcohol, cocaine abuse
- Diseases associated with saccular aneurysm (e.g. blood vessel disorders, Ehlers–Danlos syndrome, Marfan syndrome, polycystic kidney disease)
- Sickle cell disease
- Coagulopathies

COMPLICATIONS

- Vasospasm
  - Delayed ischemia; 4–11 days after SAH
  - Blood clot lysis → release of spasmogenic substances (e.g. endothelin), ↓ production of nitric oxide → vasospasm due to smooth muscle contraction → brain ischemia
- Hydrocephalus
  - Clogging of CSF drainage
- Rebleeding
  - May occur two weeks after SAH
  - ↑ tendency in individuals with

hypertension, anxiety, seizures post-SAH
    - Associated with ↑ mortality, neurological damage
- Sympathetic hyperactivity due to ↑ intracranial pressure, SAH ("sympathetic surge") → sudden, life-threatening ↑ of blood pressure due to vasoconstriction
- ↑ plasma adrenaline levels due to sympathetic hyperactivity → arrhythmias
- Over-action of sympathetic nervous system → pulmonary vasoconstriction → ↑ capillary permeability, pressure → neurogenic pulmonary edema
- Hyponatremia
- Meningitis (irritation from presence of blood)
- Seizures

SIGNS & SYMPTOMS

- Area of brain
    - *Anterior/middle cerebral artery:* numbness, sudden muscle weakness
    - *Broca's area:* slurred speech
    - *Wernicke's area:* difficulty understanding speech
- ↑ intracranial pressure
    - *Thunderclap headache:* "worst ever" headache; may be only symptom
    - Nausea, vomiting
- Altered consciousness; coma, confusion, seizures
- *Meningeal irritation:* bleeding into subarachnoid space filled with CSF → blood degradation → irritation of meninges, development of aseptic meningitis
    - Neck pain, stiffness
    - *Positive meningeal signs:* Kernig's (pain generated by knee extension from 90°); Brudzinski's (forced neck flexion → spontaneous knee, hip flexion)
    - Photophobia
- Focal neurological signs
    - Posterior communicating artery aneurysm rupture/brain herniation due to ↑ intracranial pressure → oculomotor nerve paralysis → ipsilateral ptosis; eye pointed down, out; mydriasis, loss of pupillary light reflex
    - ↑ intracranial pressure → abducens nerve paralysis → eye pointing out → diplopia

DIAGNOSIS

DIAGNOSTIC IMAGING

Noncontrast CT scan

- Fisher scale grading
    - *Group 1:* no hemorrhage
    - *Group 2:* blood depositions < 1mm, without blood clots
    - *Group 3:* blood depositions > 1mm, with localized clots
    - *Group 4:* diffuse/lack of subarachnoid hemorrhage with extension to ventricles, brain parenchyma
- Hydrocephalus
    - "Mickey Mouse" ventricular system appearance

MRI

- Visualize arteriovenous malformations (not detected by angiography)

Digital-subtraction cerebral/CT/MR angiography

- Visualize aneurysm

LAB RESULTS

- Identify hematologic abnormalities
- *PT, aPTT:* identify coagulopathies
- ↑ troponin, if heart abnormalities present

OTHER DIAGNOSTICS

- Lumbar puncture
    - ↑ erythrocytes in all three samples
    - CSF centrifugation: yellow coloration due to erythrocytes breakage, release of heme ("xanthochromia"); positive 3–4 weeks after SAH
- Physical examination
    - Characteristic neurological presentation; fever; tachycardia; fundoscopy (optic disc swelling, retinal hemorrhages)

ECG

- ↑ QRS, QT intervals; ↓ PR intervals; U waves; dysrhythmias

TREATMENT

MEDICATIONS

- *Antihypertensive therapy:* beta-blockers; hydralazine, calcium channel blockers; ACE inhibitors
- *Intracranial pressure treatment:* osmotic, loop diuretics
- *Prior all procedures:* IV midazolam (initial treatment)
- *Vasoconstriction treatment:* calcium channel blocker (e.g. nimodipine), recombinant tissue plasminogen activator
- *Seizure treatment:* phenytoin/phenobarbital
- *Pulmonary edema treatment:* diuretics, dobutamine

SURGERY

- *Aneurysm treatment:* endovascular coiling (with aneurysm obliteration), craniotomy (with aneurysm neck clipping, coiling)
- *Vasoconstriction:* aspiration/irrigation of blood clot during clipping process, CSF drainage, transluminal balloon angioplasty
- *Hydrocephalus:* temporary/serial lumbar puncture for CSF drainage, permanent ventricular shunt, ventriculostomy

OTHER INTERVENTIONS

- Vital sign stabilization
- Intubation if comatose, heart monitoring (initial treatment)
- Keep blood pressure < 140mmHg to avoid rebleeding

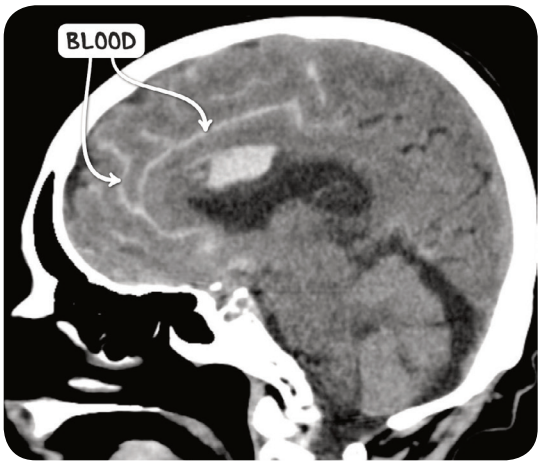

Figure 64.4 A CT scan of thea head in the sagittal plane demonstrating high signal in the sulci of the frontal lobe, consistent with a subarachnoid hemorrhage.

SUBDURAL HEMATOMA (SDH)

osms.it/subdural-hematoma

PATHOLOGY & CAUSES

- Intracranial bleeding with blood accumulation between dura mater, arachnoid membrane
- Head trauma → tearing of venous blood vessels/small cortical arteries → blood accumulation → limited blood mass expansion due to adherent dural attachments → brain tissue compression

TYPES

Acute

- Slow blood outflow into subdural space due to low pressure in bridging veins

Subacute

- Combination of fluid, clotted blood

Chronic

- Caused by minor trauma/inflammation
- More common in elderly
- Head trauma with small bleeding, dural border cell damage → inflammation, unsuccessful attempt to repair border cells with formation of granulation tissue → encapsulation; development of blood vessels within new membrane → erythrocytes, plasma exudation from leaky capillaries to encapsulated space → recurrent bleeding with expansion

CAUSES

- Head trauma (most common)
- Acceleration-deceleration (coup-contrecoup injury)
- cceleration of body → sudden stop with forwarding momentum carrying brain → impacts front of skull → backward brain movement → impacts back of skull → bridging veins tear
- Intracranial hypotension
  - ↓ CSF due to lumbar puncture/ lumboperitoneal shunt → ↑ traction of brain on surrounding structures → bridging veins tear
- Shaken baby syndrome
- Spontaneous
  - Vascular malformations
- Neurosurgical procedure complication

RISK FACTORS

- Brain atrophy elderly
  - ↑ bridging veins stretch
- Infants, alcohol abusers
  - Thinner wall of bridging veins
- Epilepsy, anticoagulant drugs, thrombocytopenia

COMPLICATIONS

- Liquefaction of granulation tissue in chronic subdural hematoma (subdural hygroma) → ↑ protein → expansion of mass due to water drawn by osmotic pressure → mass effect brain injuries
- ↑ intracranial pressure → supratentorial, infratentorial herniation of brain
- Progressive dementia in chronic subdural hematoma

SIGNS & SYMPTOMS

- Loss of consciousness after trauma/in ensuing days due to hematoma expansion
- Bleeding characteristics
  - *Hemispheric:* most common
  - *Interhemispheric:* altered consciousness, headache, hemiparesis
- Physical examination
  - Broken basilar skull: periorbital ecchymosis (raccoon eyes), retroauricular ecchymosis (Battle's sign)
  - CSF rhinorrhea/otorrhea
- Acute subdural hematoma
  - Neurological presentation in 48–72 hours

- May be comatose/awake
- Sudden, severe headache with nausea, vomiting; unequal pupils; difficulties in speech, swallowing; palsies of cranial nerves

- Subacute
  - Presents 2–14 days
- Chronic
  - Present 14 days after injury
  - Impaired cognitive skills, altered consciousness, headaches, contralateral/ipsilateral hemiparesis (depends on hematoma location), hemianopsia, optic disc swelling

DIAGNOSIS

DIAGNOSTIC IMAGING

CT scan

- *Acute:* crescent-shape hyperdense blood collection
- *Subacute/chronic:* isodense/hypodense crescentic mass

MRI

- *T2-WI (acutely):* hypointense blood clot due to deoxyhemoglobin
- *T1, T2-WI (in following weeks):* bright appearance; deoxy → methemoglobin
- *T1-WI (months later):* hypointense clot due to hemosiderin remains

MR/CT angiography

- Spontaneous SDH

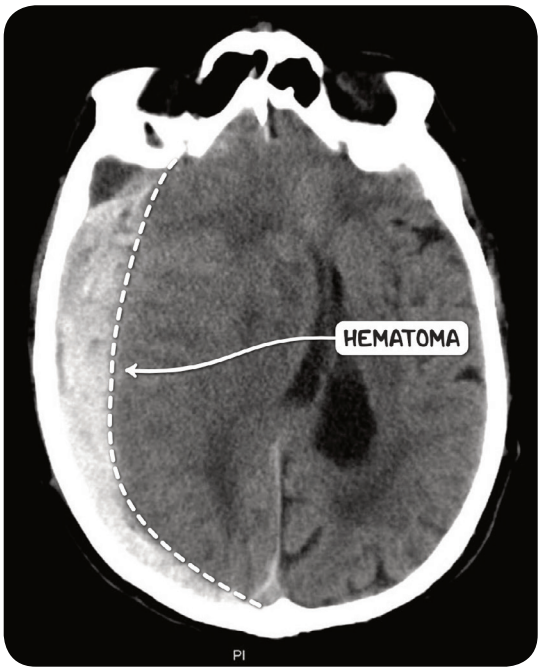

Figure 64.5 A CT scan in the axial plane demonstrating a large, right-sided, subdural hematoma. The hematoma has a classical crescentic shape.

TREATMENT

MEDICATIONS

- Diuretics
  - ↓ intracranial pressure
- Vitamin K/factor VIII inhibitor activity bypassing agent (FEIBA)/frozen plasma
  - Anticoagulation reverse; ↓ risk of hematoma enlargement; individuals undergoing surgery

SURGERY

- If clot thickness > 10mm, midline shift > 5mm, intracranial pressure > 20mmHg
  - Burr hole, craniotomy, decompressive craniectomy, blood vessel ligation

OTHER INTERVENTIONS

- Nonsurgical treatment based on Glasgow coma score (GCS); clot thickness (< 10mm); neurological examination; stable/deteriorated condition; comorbidities, associated injuries; age

TRANSIENT ISCHEMIC ATTACK (TIA)

osms.it/transient-ischemic-attack

PATHOLOGY & CAUSES

- Short-lasting neurological dysfunction due to transient focal ischemia, without infarction
- Blood vessel occlusion/stenosis → ↓ blood flow in affected region → neurological dysfunction

CAUSES

- *Adults:* thrombosis, hypoperfusion, emboli
- *Children:* congenital heart defects with thrombosis, coagulopathies, idiopathic progressive arteriopathy of childhood (Moyamoya disease)

RISK FACTORS

- More common in black individuals of African descent who are biologically male; ↑ risk with age
- Family history, hypertension, diabetes, obesity, obstructive sleep apnea, ↑ low-density lipoprotein (LDL), ↓ high-density lipoprotein (HDL), atherosclerosis, cocaine abuse, smoking

COMPLICATIONS

- Recurrent TIA
- Ischemic stroke

SIGNS & SYMPTOMS

- *Duration:* few minutes to one hour

↓ flow in large arteries (few minutes)

- Numbness/paresis
  - Face, cheek, tongue, arm, hand, leg
- Aphasia
  - If dominant hemisphere affected
- Hemispatial neglect
  - If nondominant hemisphere affected
- Distal vertebral artery
  - Dizziness, difficulty speaking, double vision
- Mid-basilar artery
  - Dizziness, paresis affecting both legs/arms

Embolic TIA (> one hour)

- Anterior cerebral circulation
  - Symptoms depend on blood vessel lodged
  - *Middle cerebral artery:* contralateral hemiplegia; aphasia if dominant hemisphere; hemispatial neglect if nondominant
  - *Branches of middle cerebral artery:* numbness/motor function loss; face, arm, leg
  - *Ophthalmic artery:* amaurosis fugax; transient monocular/binocular vision loss
- Posterior cerebral circulation
  - Dizziness, focal hearing loss, speech difficulties, double vision, hemi/quadrantanopia, face/body numbness
  - *Basilar artery:* thalamus, subthalamus, medial midbrain, reticular activating system → stupor/coma

DIAGNOSIS

DIAGNOSTIC IMAGING

CT/MR/conventional catheter angiography

- Occlusion within blood vessel

Diffusion-weighted MRI

- Ischemic regions corresponding to neurologic symptomatology
- Changes seen within first few hours of symptoms

Perfusion-weighted MRI

- ↓ tissue blood flow

Neck Doppler ultrasound

- Evaluate carotid stenosis

LAB RESULTS

- Hypoglycemia, hyponatremia, thrombocytosis: rule out conditions that mimic TIA

OTHER DIAGNOSTICS

- See mnemonic
  - *ABCD2 score:* evaluate risk for possible ischemic stroke (can occur two days after TIA)

MNEMONIC: ABCD2

Evaluating ischemic stroke risk

Age
Blood pressure
Clinical features
Duration of symptoms
Diabetes

TREATMENT

MEDICATIONS

- Antiplatelet (noncardioembolic TIA)
  - Aspirin/extended-release dipyridamole/aspirin + clopidogrel
- Anticoagulation
  - *Atrial fibrillation:* low-molecular-weight heparin
  - *Heart thrombus:* in acute myocardial infarction/rheumatic mitral valve; warfarin + direct acting oral anticoagulants (e.g. apixaban)
- Diuretics, angiotensin-converting enzyme (ACE) inhibitors
  - Blood pressure control
- Statins
  - Cholesterol management

SURGERY

- Same side carotid stenosis/TIA
  - Carotid endarterectomy

OTHER INTERVENTIONS

- Mediterranean diet